(Srinivasula, SM., Ahmad, M., Lin, JH., Poyet, JL., Fernandes, Alnemri, T.,Tsichlis, PN., Alnemri, ES, J. Biol, Chem. 18., 274(25):17946-54, (1999)).—

Please amend the paragraph beginning on page 6, line 34 and continuing onto page 7 with the following amended paragraph:

-- The transcription factor NF-kB is sequestered in an inactive form in the cytoplasm as a complex with its inhibitor, IkB, the most prominent member of this class being IkBa (Inhibitor of nuclear factor KappaB Alpha). A number of factors are known to serve the role of stimulators of NF-kB activity, such as, for example, TNF. After TNF exposure, the inhibitor is phosphorylated and proteolytically removed, releasing NF-kB into the nucleus and allowing its transcriptional activity. Numerous genes are upregulated by this transcription factor, among them IkBa. The newly synthezised IkBa protein inhibits NF-kB, effectively shutting down further transcriptional activation of its downstream effectors. However, as mentioned above, the IkBa protein may only inhibit NF-kB in the absence of IkBa stimuli, such as TNF stimulation, for example. Other agents that are known to stimulate NF-kB release, and thus NF-kB activity, are bacterial lipopolysaccharide, extracellular polypeptides, chemical agents, such as phorbol esters, which stimulate intracellular phosphokinases, inflammatory cytokines, IL-1, oxidative and fluid mechanical stresses, and Ionizing Radiation (Basu, S., Rosenzweig, K, R., Youmell, M., Price, B, D, Biochem, Biophys, Res, Commun., 247(1):79-83, (1998)). Therefore, as a general rule, the stronger the insulting stimulus, the stronger the resulting NF-kB activation, and the higher the level of IkBa transcription. As a consequence, measuring the level of IkBa RNA can be used as a marker for antiapoptotic events, and indirectly, for the onset and strength of pro-apoptotic events.--